



Approach to Acute renal failure

Dr. Munawar Mohsen Almajnoni, MD

Consultant cardiologist

Chairman of medicine department

PMAH



Objective

- Definition of ARF
- Epidemiology
- Etiology of ARF
- Management of ARF
 - Diagnosis of ARF
 - Treatment of ARF



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Epidemiology

- It occurs in
 - 5% of all hospitalized patients and
 - 35% of those in intensive care units
- Mortality is high:
 - up to 75–90% in patients with sepsis
 - 35–45% in those without

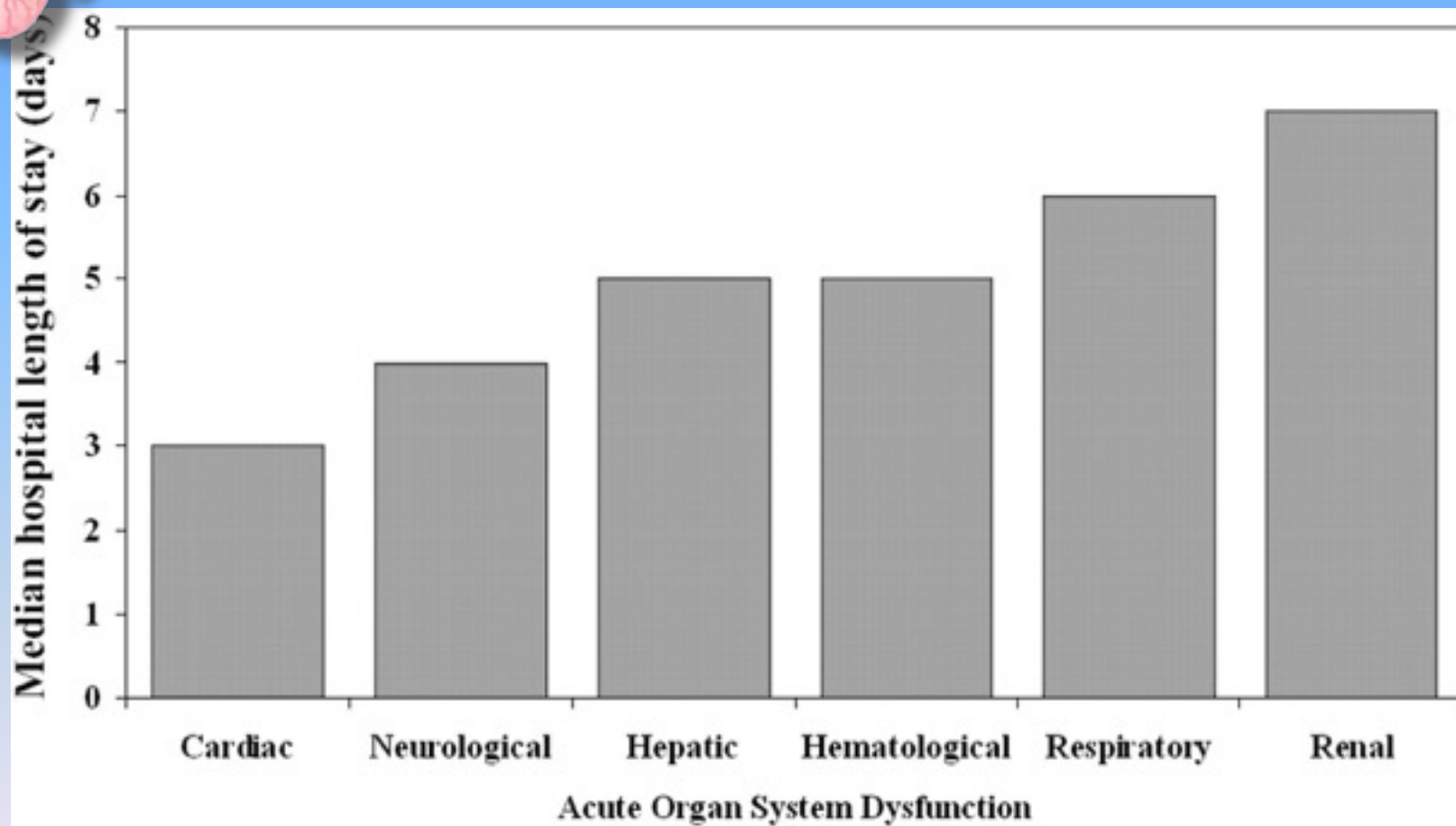


Acute renal failure (ARF) or acute kidney injury (AKI)

- Deterioration of renal function over a period of hours to days, resulting in
 - the failure of the kidney to excrete nitrogenous waste products and
 - to maintain fluid and electrolyte homeostasis
- ARF Rapid deterioration of renal function
 - (increase of creatinine of >0.5 mg/dl in <72 hrs.)
 - “azotemia” (accumulation of nitrogenous wastes)
 - elevated BUN and Creatinine levels
 - decreased urine output (usually but not always)
- Oliguria: <400 ml urine output in 24 hours
- Anuria: <100 ml urine output in 24 hours

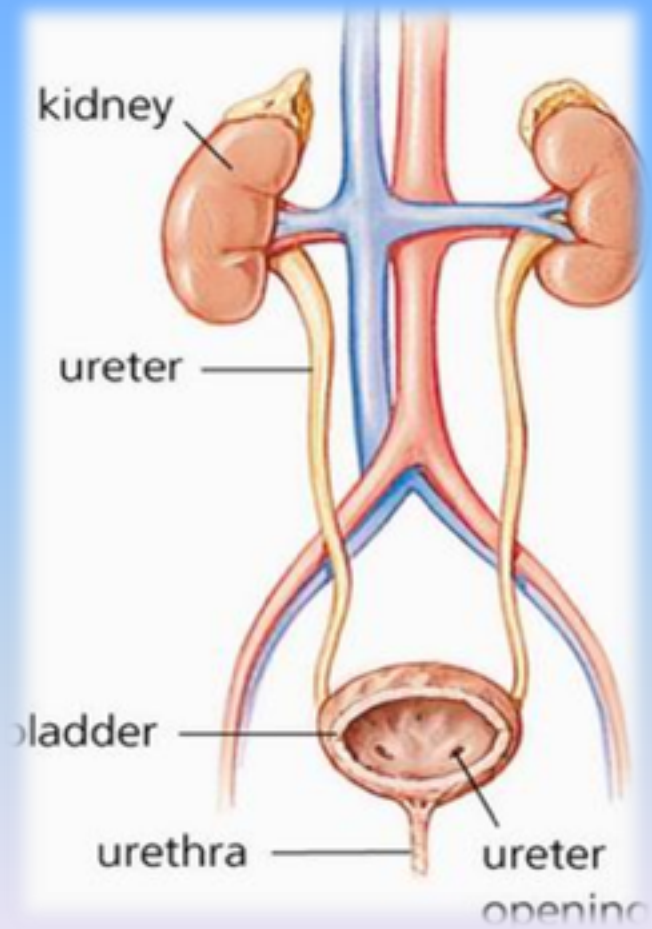


Median hospital length of stay (LOS) stratified by single acute organ system dysfunction (AOSD), including acute renal failure (ARF).



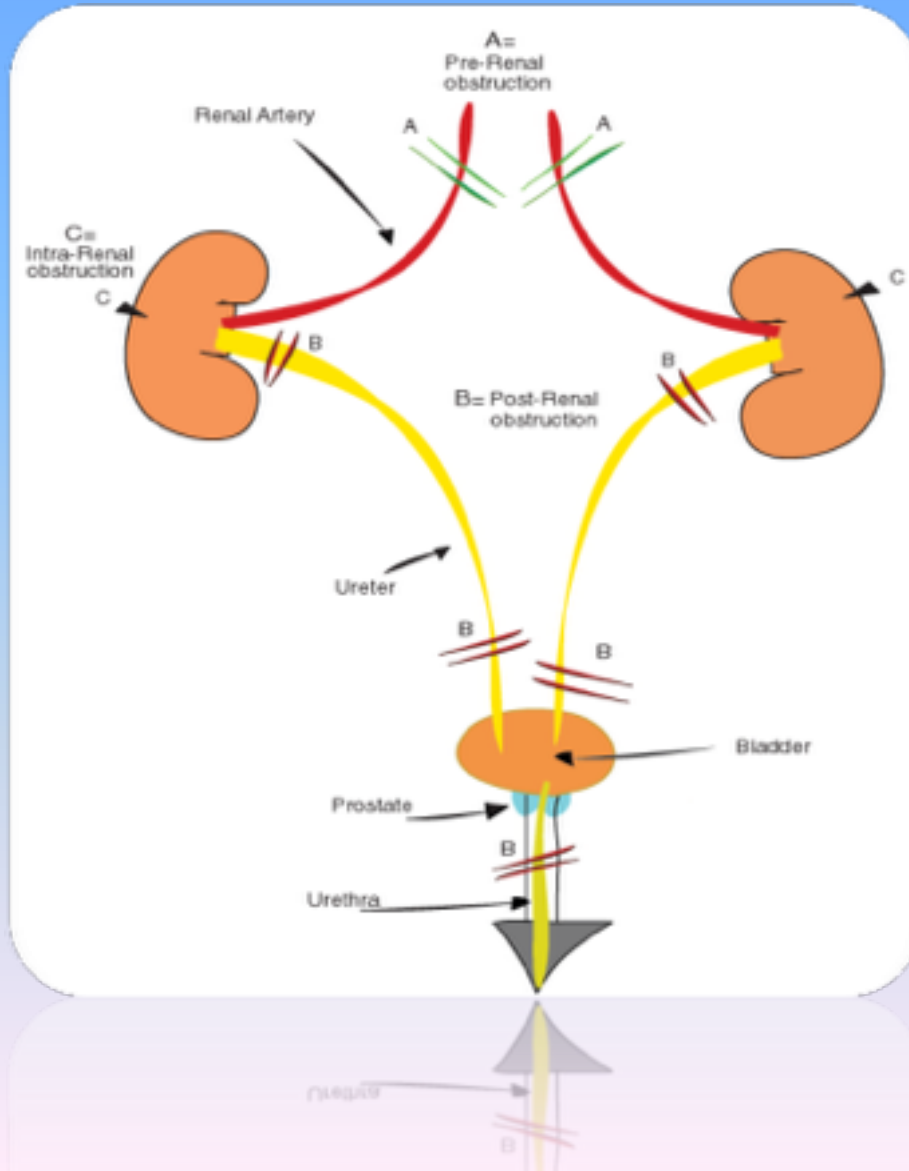


Etiology of ARF



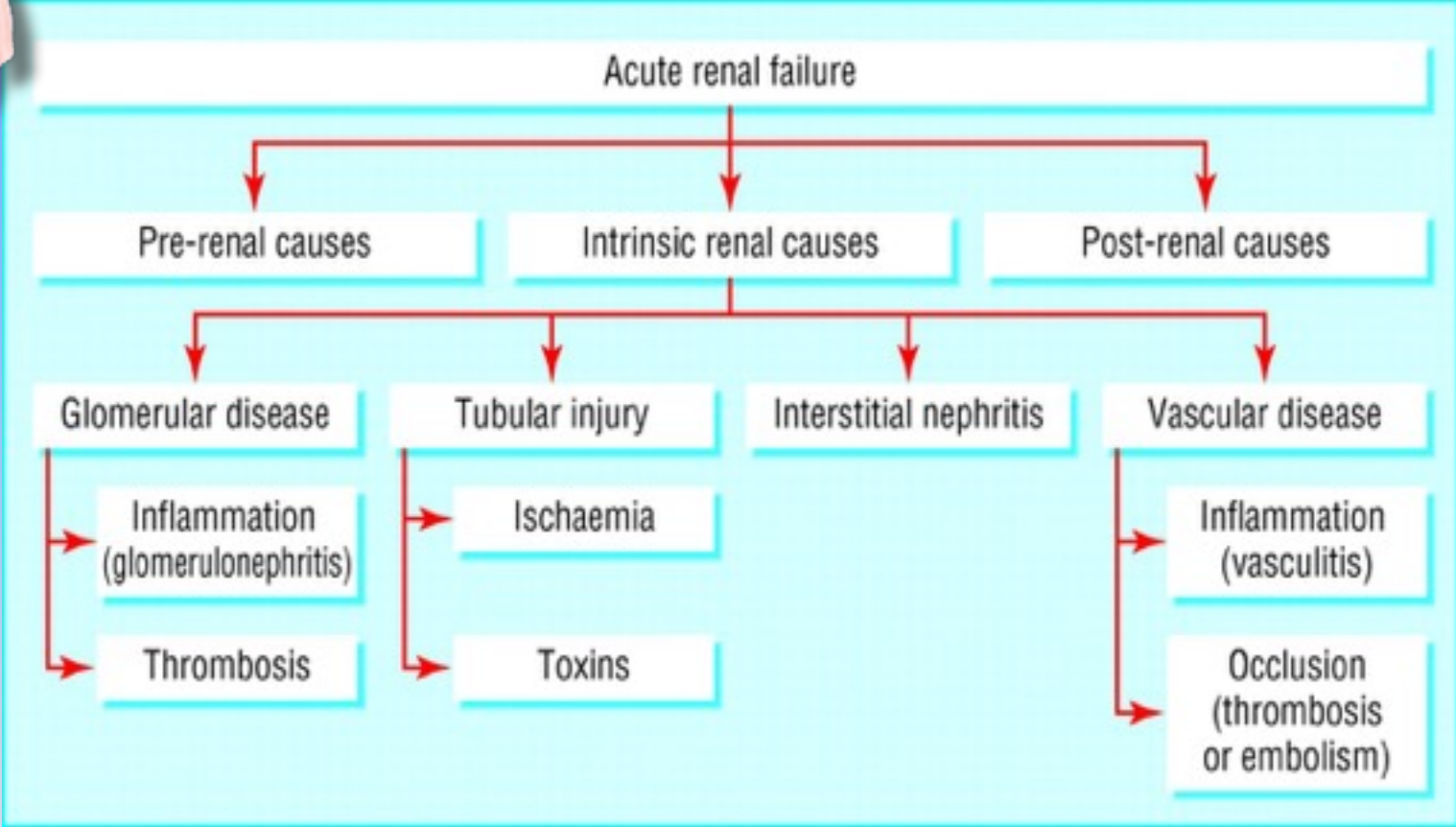


Etiology of ARF





Causes of acute renal failure





Pre-renal AKI

– Volume depletion

- Renal losses (diuretics, polyuria)
- GI losses (vomiting, diarrhea)
- Cutaneous losses (burns, Stevens-Johnson syndrome)
- Hemorrhage
- Pancreatitis

– Decreased cardiac output

- Heart failure
- Pulmonary embolus
- Acute myocardial infarction
- Severe valvular heart disease
- Abdominal compartment syndrome (tense ascites)



Post-renal AKI

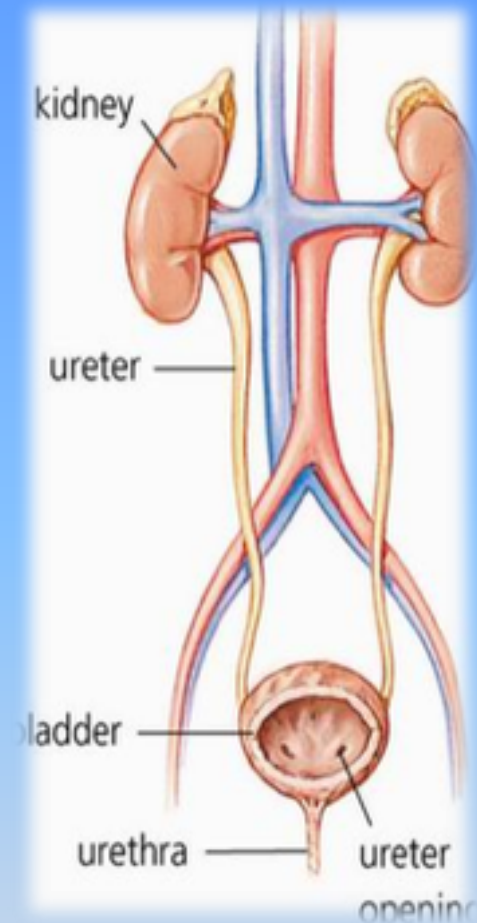
– Ureteric obstruction

- Stone disease,
- Tumor,
- Fibrosis,
- Ligation during pelvic surgery

– Bladder neck obstruction

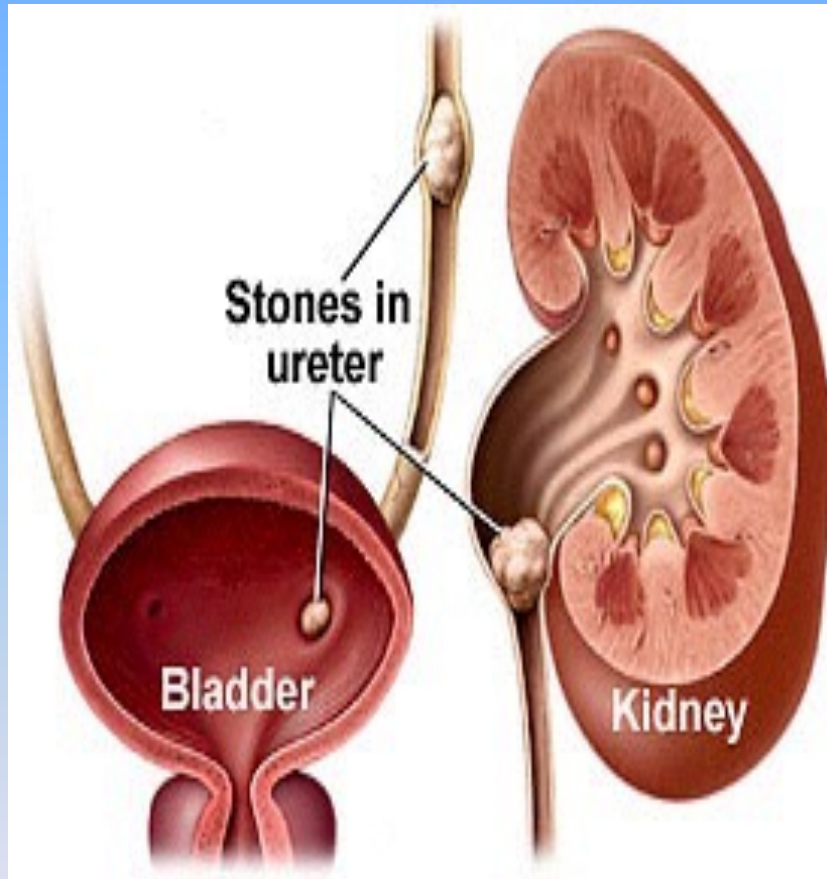
- Benign prostatic hypertrophy [BPH]
- Cancer of the prostate
- Neurogenic bladder
- Drugs (Tricyclic antidepressants, ganglion blockers,
- Bladder tumor,
- Stone disease, hemorrhage/clot)

– Urethral obstruction (strictures, tumor)





Post-renal AKI



Renal



– Glomerular

- Anti-glomerular basement membrane (GBM) disease (Goodpasture syndrome)
- Anti-neutrophil cytoplasmic antibody-associated glomerulonephritis (ANCA-associated GN) (Wegener granulomatosis, Churg-Strauss syndrome, microscopic polyangiitis)
- Immune complex GN (lupus, postinfectious, cryoglobulinemia, primary membranoproliferative glomerulonephritis)

– Tubular

- Ischemi
- Totoxic
 - Heme pigment (rhabdomyolysis, intravascular hemolysis)
 - Crystals (tumor lysis syndrome, seizures, ethylene glycol poisoning, megadose vitamin C, acyclovir, indinavir, methotrexate)
 - Drugs (aminoglycosides, lithium, amphotericin B, pentamidine, cisplatin, ifosfamide, radiocontrast agents)



Renal

– Interstitial

- Drugs (penicillins, cephalosporins, NSAIDs, proton-pump inhibitors, allopurinol, rifampin, indinavir, mesalamine, sulfonamides)
- Infection (pyelonephritis, viral nephritides)
- Systemic disease (Sjogren syndrome, sarcoid, lupus, lymphoma, leukemia, tubulonephritis, uveitis)



Clinical feature-1

- Signs and symptoms resulting from loss of kidney function:
 - decreased or no urine output, flank pain, edema, hypertension, or discolored urine
- Asymptomatic
 - elevations in the plasma creatinine
 - abnormalities on urinalysis



Clinical feature-2

- Symptoms and/or signs of renal failure:
 - weakness and
 - easy fatiguability (from anemia),
 - anorexia,
 - vomiting, mental status changes or
 - Seizures
 - edema
- Systemic symptoms and findings:
 - fever
 - arthralgias,
 - pulmonary lesions



Acute Renal Failure

Diagnosis

- Blood urea nitrogen and serum creatinine
- CBC, peripheral smear, and serology
- Urinalysis
- Urine electrolytes
- U/S kidneys
- Serology: ANA, ANCA, Anti DNA, HBV, HCV, Anti GBM, cryoglobulin, CK, urinary Myoglobin



Acute Renal Failure

Diagnosis

- Urinalysis
 - Unremarkable in pre and post renal causes
 - Differentiates ATN vs. AIN. vs. AGN
 - Muddy brown casts in ATN
 - WBC casts in AIN
 - RBC casts in AGN
 - Hansel stain for Eosinophils



Acute Renal Failure Diagnosis

- Urinary Indices;

- $$\text{FENa} = \frac{\text{UNa} \times \text{PCr}}{\text{PNa} \times \text{UCr}} \times 100$$

FENa < 1% (Pre-renal state)

- May be low in selected intrinsic cause

- » Contrast nephropathy

- » Acute GN

- » Myoglobin induced ATN

- FENa > 1% (intrinsic cause of ARF)



Acute Renal Failure

Diagnosis

- Laboratory Evaluation:
 - Scr, More reliable marker of GFR
 - Falsely elevated with Septra, Cimetidine
 - small change reflects large change in GFR
 - BUN, generally follows Scr increase
 - Elevation may be independent of GFR
 - Steroids, GIB, Catabolic state, hypovolemia
 - BUN/Cr helpful in classifying cause of ARF
 - ratio > 20:1 suggests prerenal cause



Renal failure

Differentiation between acute and chronic renal failure

	<i>Acute</i>	<i>Chronic</i>
History	Short (days-week)	Long (month-years)
Haemoglobin concentration	Normal	Low
Renal size	Normal	Reduced
Renal osteodystrophy	Absent	Present
Peripheral neuropathy	Absent	Present
Serum Creatinine concentration	Acute reversible increase	Chronic irreversible

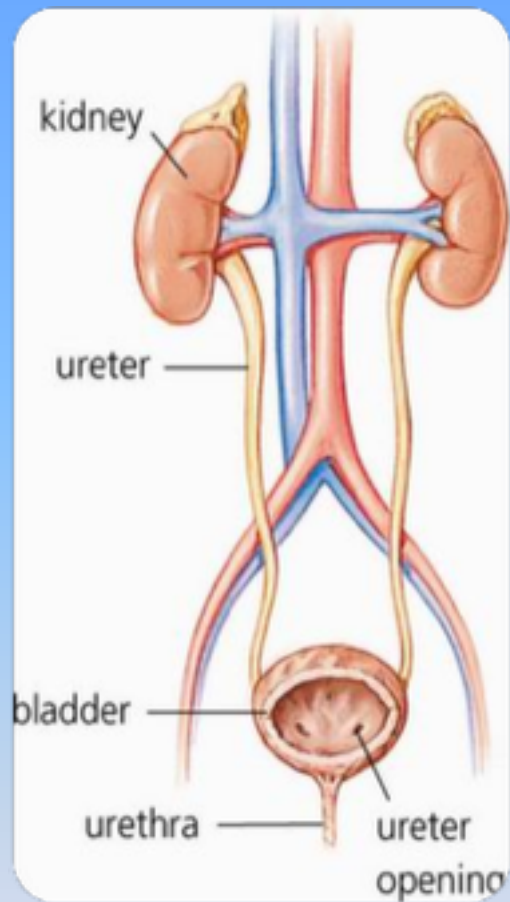


TABLE 3-3. Laboratory Tests Useful in the Diagnosis of Acute Renal Failure

Test	Favors Prerenal Disease	Favors ATN
BUN/P _{cr} ratio	>20:1	10-15:1
Rise in P _{cr}	Variable rate of rise with downward fluctuations in some patients	Progressive increase of ≥ 0.5 mg/dL per day, particularly in oliguric patients
Urinalysis	Normal or near normal; hyaline casts may be seen but are not an abnormal finding	Many granular casts with renal tubular epithelial cells and epithelial cell casts
U _{osm}	>500 mosmol/kg	<350 mosmol/kg
U _{Na}	<20 meq/L	>40 meq/L
FE _{Na}	<1 percent	>2 percent



Acute Renal failure

Causes of acute renal failure

Differentiation between Pre-renal, renal and post-renal causes

Prerenal	Renal	postrenal
Hypovolaemia	Acute tubular necrosis	Bilateral ureteric
<p>Often result of renal ischaemia → death of tubular cells or direct toxic injury by endogenous chemicals such as myoglobin (from muscle → rhabdomyolysis)</p> <p>→ Integrity of tubule is destroyed obstructions, back-leakage</p>		
obstruction	(in sepsis) Tubular obstruction	



Acute Tubular Necrosis

- Most common cause of intrinsic cause of ARF
- Often multifactorial
- Ischemic ATN:
 - Hypotension, sepsis, prolonged pre-renal state
- Nephrotoxic ATN:
 - Contrast, Antibiotics, Heme proteins



Acute Tubular Necrosis

- Diagnose by history, \uparrow FE_{Na} ($>2\%$)
- sediment with coarse granular casts, RTE cells
- Treatment is supportive care.
 - Maintenance of euvolemia (with judicious use of diuretics, IVF, as necessary)
 - Avoidance of hypotension
 - Avoidance of nephrotoxic medications (including NSAIDs and ACE-I) when possible
 - Dialysis, if necessary
- 80% will recover, if initial insult can be reversed



Contrast nephropathy

- 12-24 hours post exposure, peaks in 3-5 days
- Non-oliguric, FE Na $<1\%$!!
- RX/Prevention: 1/2 NS 1 cc/kg/hr 12 hours pre/post
- Mucomyst 600 BID pre/post (4 doses)
- Risk Factors: CKD, Hypovolemia ,DM,CHF



Rhabdomyolysis

- Diagnose with \uparrow serum CK (usu. $> 10,000$), urine dipstick (+) for blood, without RBCs on microscopy, pigmented granular casts
- Common after trauma (“crush injuries”), seizures, burns, limb ischemia occasionally after IABP or cardiopulmonary bypass
- Treatment is largely supportive care. With IVF



Acute Glomerulonephritis

- Rare in the hospitalized patient
- Diagnose by history, hematuria, RBC casts, proteinuria (usually non-nephrotic range), low serum complement in post-infectious GN), RPGN often associated with anti-GBM or ANCA
- Usually will need to perform renal biopsy

Atheroembolic ARF

- Associated with emboli of fragments of atherosclerotic plaque from aorta and other large arteries
- Diagnose by history, physical findings (evidence of other embolic phenomena--CVA, ischemic digits, “blue toe” syndrome, etc), low serum C3 and C4, peripheral eosinophilia, eosinophiluria, rarely WBC casts
- Commonly occur after intravascular procedures or cannulation (cardiac cath, CABG, AAA repair, etc.)



Acute Interstitial Nephritis

- Usually drug induced
 - methicillin, rifampin, NSAIDS
- Develops 3-7 days after exposure
- Fever, Rash , and eosinophilia common
- U/A reveals WBC, WBC casts, + Hansel stain
- Often resolves spontaneously
- Steroids may be beneficial (if Scr>2.5 mg/dl)



Acute Renal failure

Causes of acute renal failure

Changes during acute renal failure

Hyperkalaemia (→ ECG abnormalities)

decreased bicarbonate

In many cases kidney can recover from acute renal failure

The function has to be temporarily replaced by dialysis

disturbed fluid or electrolyte homeostasis must be balanced

primary causes like necrosis, intoxication or obstruction

must be treated

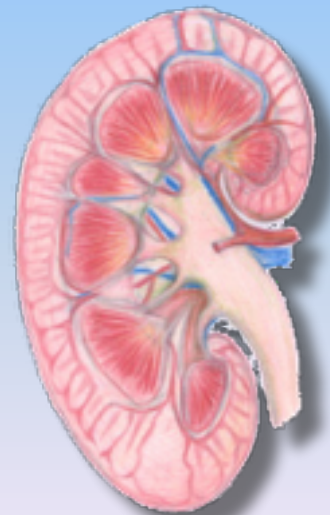


Treatment of AKI

- Optimization of hemodynamic and volume status
- Avoidance of further renal insults
- Optimization of nutrition
- If necessary, institution of renal replacement therapy

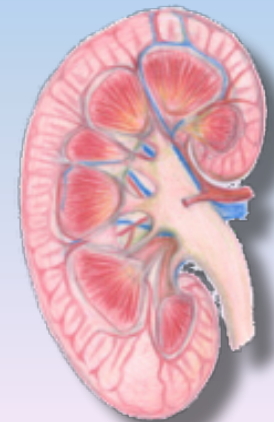
Indication for renal replacement therapy

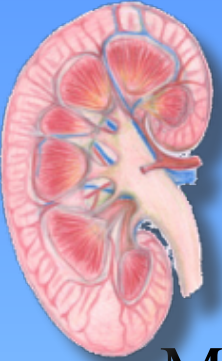
- Symptoms of uremia (encephalopathy,...)
- Uremic pericarditis
- Refractory volume over load
- Refractory hyperkalemia
- Refractory metabolic acidosis



Case-1

- 63 yrs. old women with Hx of long standing
 - DM II and HTN (20 years)
- C/O muscle aches and pain for 2 weeks
 - No Hx of nausea, vomiting and diarrhea
 - Seen 3 days before at private clinic
 - SCr 139 ALY 160 AST 83 U/A +3 glucose, +1 protein





Case-1

- Medications list:
 - Bisoprolol, Irbesartan, Simvastatin, and Gemfibrozil
- On Ex:
 - ill looking, Bp 140/90, P =105/min, O2 sat 95% on room air, JVP 3-4 cm ASA
 - No L.L oedema
 - Muscle tenderness with normal power
 - Chest: normal
 - CVS : normal S1 and S2 no murmurs



Differential diagnosis of acute renal failure

Is this acute or chronic renal failure?

- History and examination
- Previous creatinine measurements
- Small kidneys on ultrasound (except diabetes)

Has obstruction been excluded?

- Complete anuria
- Palpable bladder
- Renal ultrasound

Is the patient euvolaemic?

- Pulse, JVP/CVP, postural blood pressure, daily weights, fluid balance
- Disproportional increase in urea:creatinine ratio
- Urinary sodium concentration (unless on diuretics)
- Fluid challenge

Does evidence of renal parenchymal disease exist (other than ATN)?

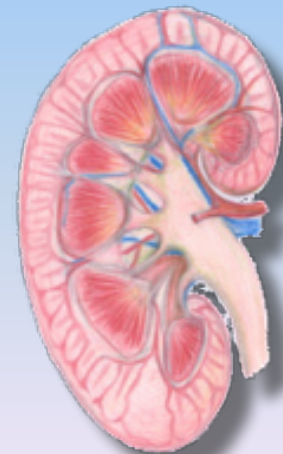
- History and examination (systemic features)
- Urine dipstick and microscopy (red cells, red cell casts, eosinophils, proteinuria)

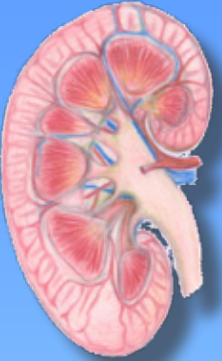
Has a major vascular occlusion occurred?

- Atherosclerotic vascular disease
- Renal asymmetry
- Loin pain
- Macroscopic haematuria
- Complete anuria

Case-1

- SCr 350
- CK very high
- K =5.2
- U/A +3 protein,+3 Hb
- U/S kidney

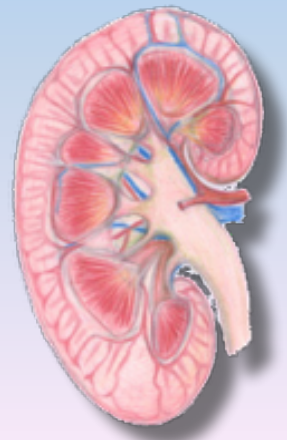




Diagnosis and Treatment

Case -2

- 70 years old male
- C/O Vomiting blood for 1 day
- On Ex:
 - Bp 120/80 mmHg ,P=100/min JVP 4cm
- Lab:
 - SCr 80, urea 11
- Diagnosis?



Pre-renal AKI

- History:
- Physical examination
 - Volume status
 - Blood pressure, Pulse, JVP
 - Urine out put
- Investigation:
 - SCr, urea
 - Urine analysis
 - Urine electrolytes

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